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Review article

Lung health in era of climate change and dust storms

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ABSTRACT

Dust storms are strong winds which lead to particle exposure over extensive areas. These storms influence air quality on both a local and global scale which lead to both short and long-term effects. The frequency of dust storms has been on the rise during the last decade. Forecasts suggest that their incidence will increase as a response to the effects of climate change and anthropogenic activities.

Elderly people, young children, and individuals with chronic cardiopulmonary diseases are at the greatest risk for health effects of dust storms. A wide variety of infectious and non-infectious diseases have been associated with dust exposure. Influenza A virus, pulmonary coccidioidomycosis, bacterial pneumonia, and meningococcal meningitis are a few examples of dust-related infectious diseases. Among non-infectious diseases, chronic obstructive pulmonary disease, asthma, sarcoidosis and pulmonary fibrosis have been associated with dust contact. Here, we review two molecular mechanisms of dust induced lung disease for asthma and sarcoidosis. We can also then further understand the mechanisms by which dust particles disturb airway epithelial and immune cells.

1. Introduction

1.1. Dust storms

Climate change has created a wide array of major fluctuations in the environment. Variations in rainfall patterns, rising sea levels, and increased severe weather phenomena such as droughts, floods and dust storms, all have an undeniable implication on human health (Mirsaeidi et al., 2016).

Dust storms, defined as an atmospheric phenomenon triggered by numerous small particles evenly distributed in the air, provide a vital role on Earth (2, 3). Such storms have lasting air quality impact on both a local and global scale in the short and long term (Duncan Fairlie et al., 2007). The majority of dust storms arise from arid and semi-arid regions, frequently referred to as drylands. Drylands are typically formed in the subtropics, where the sinking branch of Hadley cell brings warm and dry air to the land's surface. In meteorology, Hadley cell is defined as a large-scale atmospheric convection in which air rises at the equator and sinks at medium latitudes. Since the air near Earth's surface is dry, the land areas of these regions (approximated as 30°N and 30°S of equator) are the location of world's major deserts (Fig. 1).

These areas are noted to generally be in low-lying terrains (< 1800 m of elevation), with high aerosol index and a low average annual rainfall (Ghio et al., 2014; Goudie and Middleton, 2006). The association between drylands and dust storms is explained by the fact that desiccated, unconsolidated substrates with scarce vegetation cover permit turbulent winds to easily raise particles from the surface (Middleton, 2017). Drylands constitute roughly 40% of the world surface, and are home to approximately 30% of the world's population (Safriel et al., 2005). It is important to note that the impact of dust storms is able to exceed these boundaries, transporting particles out to areas thousands of kilometers away from their origins (Claiborn et al., 2000; Kim et al., 2005). In this paper, we review the impact of particle inhalation and its association with pulmonary diseases, with a specific focus on asthma and sarcoidosis.

1.1.1. Areas of origin

The most important dust source along the dust belt is located in the Sahara-Sahel region in Northern Africa. This region is home to the largest source of atmospheric desert dust on the planet, contributing to almost half of the world's total dust budget. Meanwhile, China and Central Asia contribute around 20%; followed by Arabia and Australia.

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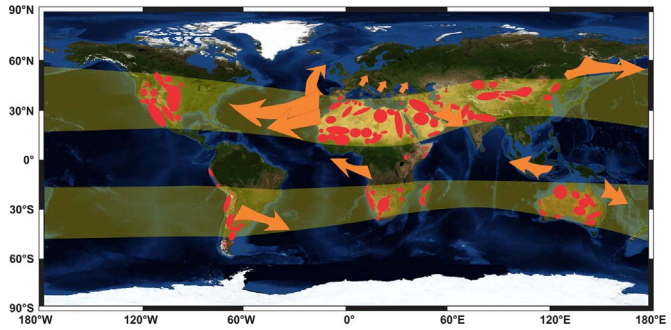

Fig. 1. Schematic map of dust belt and dust transportation pathways. Dust source regions (red) are based on daily measurements of dust optical depth using Moderate Resolution Imaging Spectroradiometer Deep Blue (MODIS DB) Level 2 (Ginoux et al., 2012). The orange arrows denote the dust transport paths over the ocean based on the composite of monthly mean of Total Ozone Mapping Satellite (TOMS) absorbing aerosol index (AAI) (Maher et al., 2010). The pale yellow bands show the approximate locations of the global dust belt. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

Both Southern Africa and the North and South America contribute a lesser yet significant amount, calculated at no more than 5% of the planet's dust production. (Miller et al., 2004) The latter estimates are based on models, as actual chronological and geographical measurements are sporadic (Middleton, 2017). In the United States, data from the National Weather Service indicates that the largest number of dust storms occur in the Arizona, California, Washington and Nevada during the spring and summer months. July has been documented as the month with the highest activity of dust storms. These storms occur more frequently in the early afternoon hours (Crooks et al., 2016). To further quantify the effects of dust exposure on Earth, scientists have begun to use satellite data as a means to determine regional air pollution and identify areas of high dust exposure. This technology is increasingly being used to estimate airborne dust around the world, especially in areas where direct measurements are unavailable (Chudnovsky et al., 2017).

The temporal and spatial production of dust is not constant. The Sahara Bodélé Depression, the world's largest desert dust source, is constantly active. However, it has been reported that an increase in activity (affecting West Africa) occurs between the months of October and April. The Middle East is affected particularly between May and August when the northwestern winds of Shamal increase in frequency (Middleton, 2017; Yu et al., 2016). Desiccation of lakes caused by poor water management is seen in Central Asia (Issanova et al., 2015) and the Middle East (AghaKouchak et al., 2015). Land surface erosion, like that seen in the North American Great Plains (McLeman et al., 2014), and deforestation are clear examples of how human activities have made substantial contributions to the creation of new drylands and new potential sources of dust.

1.1.2. Dust transport

Emerging research show that particles can travel long distances through the troposphere. For example, from the Taklamakan Desert in Northwest China and from North Africa can traverse the Pacific and Atlantic Oceans, respectively. These particles can be found in such far-reaching places as North America (McKendry et al., 2001; Pourmand et al., 2014) and Greenland (Bory et al., 2003). In 2007, particles traveled around the globe in a period of just 13 days (Uno et al., 2009). Dust hazards from the Gobi Desert in China have been observed in many cities, including Beijing (Liu et al., 2014), Seoul, and Tokyo (Kashima et al., 2016). Saharan dust has been shown to move across the Atlantic Ocean reaching the Amazon (Swap et al., 1992), the Andes (Boy and Wilcke, 2008) and many other locations in the Americas (Bozlaker et al., 2013; Pourmand et al., 2014). They have also been identified to head northbound into Europe (Varga, 2012) and the Arctic (Barkan and Alpert, 2010). The Sahara is known as a principle source of dust deposition in the Mediterranean Sea. Saharan dusts have been seen in many cities including Madrid, Barcelona, Rome and Athens (Salvador et al., 2014). Mid-East dust has been shown to reach all over the region including Iran (Givehchi et al., 2013) and India (Badarinath et al., 2010). Global dust transport paths over the ocean are shown in Fig. 1.

1.1.3. Dust composition

Dust composition is influenced by a wide range of natural and anthropogenic factors. Particle size and conformation are largely determined by the structure and composition of source rocks and physical and chemical weathering processes. Additionally, wind velocity and atmospheric conditions can greatly influence the dust mixing process during transportation. Particles found near dust storms are comprised of weathering resistant minerals such as quartz, titanium bearing minerals and zircon, those found at long distances from the storm's origin are comprised of clay minerals and phyllosilicates. Particles generally consist of silicon dioxide (SiO_2), aluminum oxide (Al_2O_3), iron (Fe_2O_3) and titanium (TiO_2) oxides. Other common particles are calcium (CaO) and magnesium oxide (MgO), and oxides of sodium and potassium (Na_2O and K_2O). Based on the composition of source rock and mineralogy of dust, an array of trace elements such as zirconium (Zr), strontium (Sr), rubidium (Rb) and rare earth elements (REEs) are also present. Desert dust also contains large amounts of evaporated minerals (salt), organic content, pathogens and anthropogenic pollutants (heavy metals, pesticides, sulfate, nitric acid, polycyclic aromatic carbons) in its matrix and/or surface (Middleton, 2017). Mineralogy, chemical composition and particle size of mineral dust are widely used to track the dust particles to their source regions (Coudegaussen et al., 1987; Sarnthein et al., 1981). Ultimately, utilizing isotopic tracers (Sr, Nd and Hf) seems to provide more reliable information about dust deposit origins and source contributions (Grousset et al., 1988; Pourmand et al., 2014). The Nd-Sr composition of the world's major dust sources is shown in Fig. 2.

Among dusts, many microorganisms including bacteria, fungi, spores have been described. Even viruses have been described as a part of the microorganisms of those particles (Chen et al., 2010; Griffin, 2007; Maki et al., 2010). These have survived for long periods of time as demonstrated in analysis of dust collected by Charles Darwin from over 150 years ago (Gorbushina et al., 2007). Some of these pathogens, especially those in spore forms (for examples *Bacillus*) have been described to endure UV and gamma-ray radiation, low temperatures and desiccation instigated by transportation at high altitudes (Saffary et al., 2002).

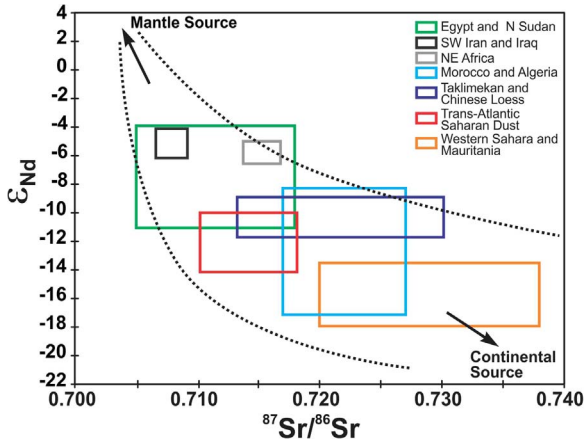

Fig. 2. Radiogenic Sr and Nd isotopes in aerosols from the world's source regions. Colored boxes represent ranges of values measured in surficial samples of North Africa, East Asia and Middle East (Abouchami et al., 2013; Chen et al., 2007; Chen and Li, 2013; Scheuvens et al., 2013) as well as Trans-Atlantic Saharan Dust particles collected in Bahamas (Pourmand et al., 2014).

1.1.4. Dust size

Mineral particles have a wide range of sizes, which are classified according to their particulate aerodynamic matter (PM) diameter size in micrometers (μm). Commonly, the largest are coarse particles ranging from PM_5 to PM_{10}, while the smaller or fine particles are $PM_{2.5}$. Even smaller, ultrafine, particles size $PM_{0.1,}$ have been identified. The largest portion of particles in desert storms tends to be coarse, mainly of silt-sized (4–62.5 μm) and clay-sized (4 μm) particles (Engelbrecht et al., 2009; Tam et al., 2012). Most Middle East generated dusts are sized between 2 and 20 μm, with 85% being less than PM_{10} (Middleton, 2017).

The PM_{10} dust concentrations greater than 1000 $\mu m/m^3$ are common in dust storms (Middleton, 2017), surpassing 15,000 $\mu m/m^3$ in severe events (Leys et al., 2011), a number that clearly exceeds the World Health Organization (WHO) recommendations of safe levels of PM_{10} (mean of 50 $\mu m/m^3$/24 h in air quality guidelines) (Organization, 2006). The Korean Meteorological Administration has developed a public health notification system based on PM_{10} concentrations over a period of 2 h in the event of dust storms. A dust advisory is issued when concentrations exceed 400 $\mu m/m^3$, while levels above 800 $\mu m/m^3$ triggers a dust warning (Asian Dust, 2016).

2. Discussion

2.1. Dust particle inhalation and lung disease

A variety of health effects have been related to dust storms. The ease of accessibility for particles to enter through the respiratory system predisposes the lungs to potentially harmful inorganic and organic components. Certain populations are at greater risk of the health effects of dust storms. Elderly people are predisposed due to dusts' effects by aging's natural effective immunologic decline and deterioration. Young children, whose immune systems are still in the process of maturing their respiratory responses to antigens (Jakubiak-Lasocka et al., 2015), as well as individuals suffering from chronic cardiopulmonary diseases, are also considered to have higher risk (Jimenez et al., 2010).

The impact of dust storms on health is principally determined by the particle size. Moreover, the size is closely related to its toxicity (Huang et al., 2014). The respiratory tract is the main destination of inhaled particles. Larger particles usually are cleared by the mucociliary system either in the nose or throat. Inhaled particles less than PM_{10} do not evade these filters, and subsequently can reach bronchioles or even alveoli (Pinkerton et al., 2000; Sandstrom and Forsberg, 2008). While most PM_{10} settle in the respiratory tract, fine particles ($PM_{2.5}$) account for 96% of the particles spotted in the lung parenchyma, penetrating into gas exchange regions of the lung (Churg and Brauer, 1997). Therefore, particles sized $PM_{2.5}$ further provoke a series of airway damages as it may trigger apoptosis, autophagy, and a series of other oxidative stress mechanisms (Gualtieri et al., 2011; Huang et al., 2014). Ultra fine particles ($PM_{0.1}$) may actually pass through the lungs and access other organs through the circulatory system (Brook et al., 2010; Martinelli et al., 2013; Xu et al., 2008). The mechanism of toxicity of ultra-fine particles remains unclear and needs further investigation (Terzano et al., 2010).

2.1.1. Infections

A wide array of pathogenic and non-pathogenic organisms are found in desert dust. Dust serves as a carrier promoting both colonization and infection upon inhalation. This has been explained under the premise that particles mount an inflammatory response in the respiratory airways, particularly in patients with pulmonary disease (Lundborg et al., 2001). Although the virulence factors of many of the microorganisms traveling with dust is not well understood, it has been suggested that exposure to high temperatures ($> 39.5\,^{\circ}C$) and particles inhalation infers a significantly elevated risk in the development of pneumonia. This combination alters the function of the host's immune system, which in turn facilitates bacterial infections (Jusot et al., 2016). A stable, innocuous nasopharyngeal bacterial colonization may progress into a pneumonia and into even deeper infections, such as meningitis. *Neisseria meningitidis* has been suggested to proliferate in the setting of high dust loads, further assisted by persistent low humidity which may affect mouth immune defenses (Moore, 1992). An iron-rich dust environment (Noinaj et al., 2012) and a co-occurrence of viral respiratory infections (Mueller et al., 2008) may lead to the penetration of inhabiting pathogens from the respiratory mucosa into blood and tissue after exposure to dust storms (Kang et al., 2002). Temporal correlation between increased dust events and incidence of meningitis is witnessed in West Africa (Agier et al., 2013). Moreover, an increase in viral pneumonia incidence is observed subsequent to Asian dust events (Cheng et al., 2008; Kang et al., 2012). An increased incidence of Influenza A infection has been reported during dust storm days in Taiwan (Chen et al., 2010).

Fungal infection is another concern during dust storms. Coccidiomycosis is a disseminated infection resulting from the inhalation of *Coccidioides immitis* airborne spores found in soil. It is suggested to be related to dust storms occurring in the southwest United States (Goudie, 2014; Stockamp and Thompson, 2016). This fungal disease, which affects mainly the lungs and can be potentially fatal, shows a bimodal seasonality that coincides with the drier and dustier months in California and Arizona (Comrie, 2005). This so called "Valley Fever", reports an annual incidence of around 150,000 cases in the US (Anderson, 2013; Zender and Talamantes, 2006).

The above examples may explain the reason for increasing mortality in affected regions just after dust storms. Perez et al. (2008) found daily increase of 10 $\mu g/m^3$ of $PM_{10-2.5}$ was associated with a daily increase of mortality by 8.4% during Sahara dust storms in Barcelona. Kunzli et al. (2000) found similar results in a larger European dataset.

2.1.2. Pneumoconiosis, desert lung syndrome

People inhabiting places with increased exposure to desert dust have a higher risk of developing pneumoconiosis (Ghio et al., 2014). A significant amount of dust storms contain silt-sized quartz (sediment particles of 0.004–0.06 mm), which if inhaled over prolonged periods of time may cause desert lung disease or non-occupational silicosis (Castranova and Vallyathan, 2000; Goudie, 2014). Alveolar macrophages consume silica elements and subsequently mount an inflammatory reaction, which can lead to lung fibrosis (Hamilton et al., 2008). This phenomena particularly affects the elderly population in areas of Northwest China and Northern India (Norboo et al., 1991; Saiyed et al., 1991). It is also suggested that silicosis may play a major role in the high prevalence of tuberculosis found in this affected population (Mathur and Choudhary, 1997). Although the immune response to silica is not well understood, animal models implicated the role of T helper (Th) 1 and Th2 cells in the pathogenesis of silicosis (Davis et al., 1999; Garn et al., 2000). In addition, innate immune response may play a role in silicosis (Beamer et al., 2010).

2.1.3. Chronic respiratory diseases: asthma, Chronic Obstructive Pulmonary Disease (COPD)

According to ISAAC (The International Study of Asthma and Allergies in Childhood) and ECRHS (The European Community Respiratory Health Survey), the global prevalence of asthma has risen over time (Pearce et al., 2000). Although the increased frequency of dust storms in the world may not be the only driver, it is an important variable to consider. In fact, dust storm affected Middle Eastern countries report the highest prevalence of asthma around the world. In 1995, Saudi Arabia reported a nationwide prevalence of asthma of 23%, indicating a significant increase from 8% in 1986 (78). Those particles carry a vast number of allergens, ranging from dust mites, pollen, anthropogenic pollutants and fungal spores (Griffin, 2007; Maki et al., 2010). These findings can further explain the association between asthma and dust exposure. Studies in Japan and Korea have identified

dust events to increase both hospitalizations and emergency room visits in pediatric populations (Kanatani et al., 2010; Lee and Lee, 2013). In Seoul, South Korea, dust events cause a 22% increase in the rate of asthma treatments within a 6-day post storm period (Lee and Lee, 2013).

COPD exacerbation hospitalizations were significantly increased in Hong Kong during instances when PM_{10} and $PM_{2.5}$ levels were found to be elevated. This is true year-round but especially during the winter season (Ko et al., 2007). These increases in morbidity are representative of the health effects coarse particles have on patients with chronic lung disease. A study from Taiwan showed a 20% increased rate of emergency visits for COPD exacerbations after each dust storm (Chan et al., 2008).

The immune system plays a vital role in the setting of desert dust exposure and chronic respiratory diseases. As expected, an adequate immune response is necessary to eliminate particles, microorganisms and allergens. However, an impaired response could mount the basis for a pathological outcome; a notion that is further worsened by the erratic immune reaction that the causal agent can cause on the host itself.

Epithelial cells in the airway serve both as a physical barrier and enhances the immune cell's response to desert dust. The activation of a variety of receptors (Toll-like receptors, C-type lectin receptors and protease-activated receptors) in these cells trigger molecular signaling mediated by pro-inflammatory cytokines (interleukin IL-6, IL-8, IL-25, IL-33). These cytokines are capable of stimulating lymphocytes, dendritic cells and granulocytes (Thacker, 2006; Wang, 2013). In asthmatic patients, cytokines are responsible for stimulating a Th2 type immune response that will further increase the production of immunoglobulin E (IgE) through IL-4 and IL-13 (Bartemes and Kita, 2012). Th2 cells also secrete IL-5, which is involved in eosinophil maturation, and IL-13 involved in mucus production and airway remodeling (Hansel et al., 2013). Th17 cells, another subset of lymphocytes, increase cytokine production of IL-17A and IL-17F and facilitate the recruitment, activation and migration of neutrophils where dust particles settled in the lung (Aggarwal and Gurney, 2002; Aujla and Alcorn, 2011). Fig. 3 illustrates the potential molecular mechanism of asthma exacerbation after dust exposure.

When considering COPD, the innate immune response, when compared with the acquired immune response, has a preponderant contribution in the progression of inflammation and remodeling of the lung (Maeno et al., 2007). In addition, Th1 and Th17 cells have regularly been found to contribute with the COPD pathogenesis (Harrison et al., 2008; Saetta et al., 1999).

2.1.4. Granulomatous disease and sarcoidosis

Sarcoidosis is a chronic multisystem disease of unknown etiology characterized by a granulomatous reaction, commonly involving the lungs (Baughman et al., 2011). It has been suggested that sarcoidosis may be related to environmental factors (Jajosky, 1998).

The best association between dust and sarcoidosis has been described in Gulf War veterans. Gulf War veterans are military members who served in active duty in Southwest Asia during the Persian Gulf War in Iraq, Kuwait, Saudi Arabia, Bahrain, Qatar, Oman, Afghanistan, or the United Arab Emirates. As shown in Fig. 1, all these countries are located in the dust belt. Smith et al. (2009) reported that the incidence of respiratory disorders in veterans who have served in the Middle East is higher than veterans deployed elsewhere, most likely due to the significant higher dust exposures. Another group of researchers found the incidence of sarcoidosis was higher in Gulf War veterans than that of the civilian population (Axelrod and Milner, 2000).

The association between zirconium (a chemical element of dust), among other metals, and granuloma formation in the lung has been reported in two previous studies (Romeo et al., 1994; Werfel et al., 1998). The biologic components of dust could be another etiology for granulomatous reaction in the lung. Both fungal cell wall agents (FCWAs) and bacterial lipopolysaccharide (LPS) contain organic dust are linked with granulomatous diseases (Newman et al., 2004; Rose et al., 1998). FCWAs and LPS stimulates peripheral blood mononuclear cells in sarcoidosis patients suggest that excessive inflammatory cytokine response may be mounted and resulting in the progression and even development of pulmonary sarcoidosis (Stopinsek et al., 2016). Fig. 4 illustrates the potential molecular mechanism of granuloma

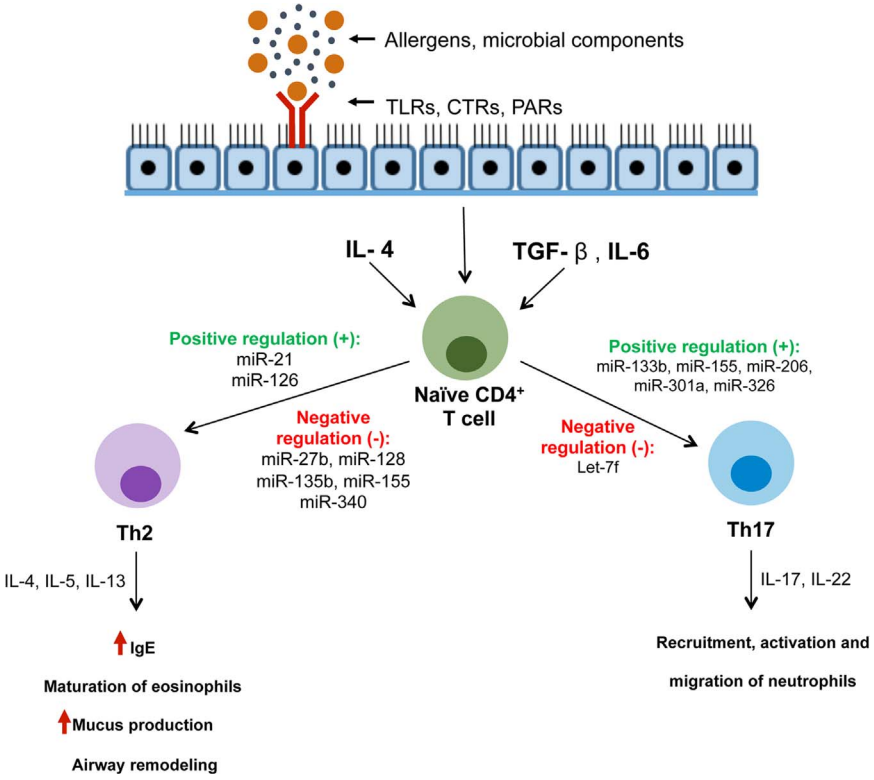

Fig. 3. Particle inhalation and asthma exacerbation. Allergens, microbial components including lipopolysaccharides (LPS) and β-glucan, and other dust particles in the air are recognized by a series of different receptors on epithelial cells, macrophages and dendritic cell in the lungs. Upon activation of these receptors, pro-inflammatory cytokines will be released triggering activation of innate lymphocytes. Lymphocytes will differentiate into Th2 and Th17 cells under the influence of cytokines and noncoding RNAs, specifically microRNAs (Sethi et al., 2013). (+) involves miRNAs that positively regulates their differentiation and (−) involves miRNAs that have a negative effect during their differentiation. This would further mediate terminal maturation of eosinophils, assist in IgE release, increase mucus production and enhance neutrophil activation, recruitment and migration. These series of events will ultimately lead to an acute asthma exacerbation.

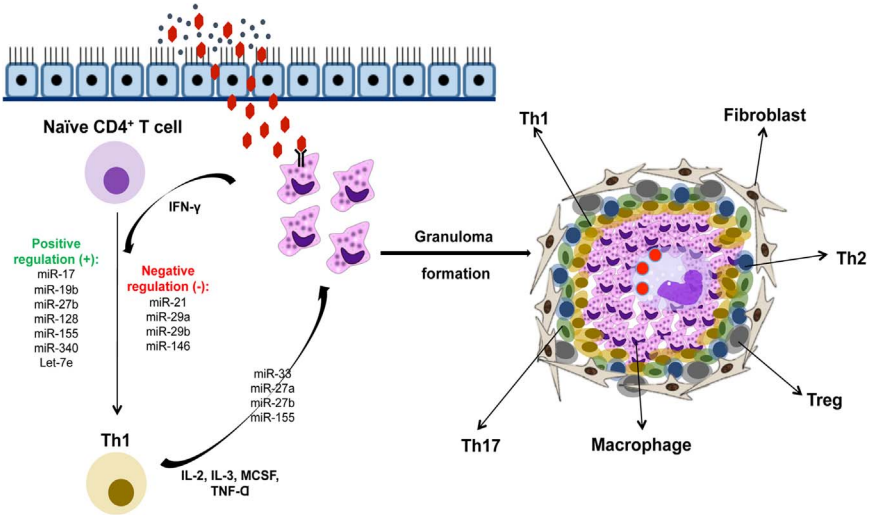

Fig. 4. Dust particle inhalation and granuloma formation. Dust and biological particles in the air enter in contact and penetrate into the human airway. Alveolar macrophages engulf dust particles initiating an immune response responsible of activating and differentiating Naïve $CD4^+$ T cells into Th1 cells through the release of inflammatory mediators and microRNAs (Jeker and Bluestone, 2013; Miyara et al., 2006). Th1 cells subsequently release multiple cytokines increasing the inflammatory response, further inducing the formation of granuloma. Some microRNAs have been implicated in the activation of macrophages, but there is no available information regarding granuloma formation in the context of sarcoidosis and its relationship with microRNA dysregulation.

formation after dust exposure in the human airways. Particles with rare earth elements (REEs), also known as mineral dust, might have the potential to instigate sarcoidosis. REEs, a set of 17 elements with similar properties that are commonly found on earth's surface, are recently gaining more attention because they have been related to the development of pulmonary diseases. Given all the above findings, we would suggest that sarcoidosis in Gulf War veterans may occur due to excessive exposure to the dust storms of the Middle East. Further investigation is warranted to assess this association.

3. Future perspective

To enhance our understanding of the pathogenesis of dust related diseases, we need more epidemiological and translational research. By understanding the pathologic interaction of dust on human airway epithelial cells, we may be able to attenuate or prevent the negative effect of dust on lung tissue.

We would propose public health officials to consider implementation of comprehensive public warning systems to reduce outdoor exposure to dust storms events, particularly for those with chronic lung diseases in the US. Satellite borne sensors and newer technologies have provided the opportunity to identify and collect larger amounts of data surrounding dust storms. This should prompt the joint cooperation of both the medical and environmental communities in the quest to better predict and understanding the health implications of dust particle exposure. To achieve these goals, we propose to develop a dust-induced disease (DID) network on national and international levels. The DID-network will be a comprehensive platform to conduct epidemiologic, clinical, and basic research in a collaborative environment involving climate change scientists, geologists, epidemiologists, microbiologists, and physician-scientists. The DID network will receive real time data regarding dust storms, particularly when high concentration of particles less than PM_{10} is achieved. Furthermore, the DID network will be capable to announce actual and accurate warnings for populations at risk, especially for patients with chronic pulmonary diseases. DID network will build the infrastructure to facilitate research projects and support funding mechanisms concerning the biological and geochemical effects of dust particles on human health.

4. Conclusions

The frequency of dust storms has increased during recent years and forecasts suggest that this will continue to rise in response to climate change and anthropogenic activities. Exposure to dust is an important risk factor for many respiratory diseases. In order to improve diagnosis and treatment of dust-induced respiratory diseases, interaction of dust with bronchial epithelial and immune cells should be further studied. It has been suggested that dust particles induce apoptosis, autophagy and oxidative stress damage in bronchial epithelial cells that may cause permanent damage in the bronchial tree. A dust alarm system could be a preventive measure to decrease exposure. Therefore, dust alarm system should be internationally implemented to advise the public to avoid exposure to dust storms.

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Authors contributions

Conception and design: M.D.S, M.M; Drafting the manuscript for important intellectual content: M.D.S, A.S.C, O.S, A.S, N.K, G.H, M.C., M.M.

Conflict of interest

Authors declare no conflict of interest to disclose.

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